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Electrocardiographic Monitoring of Myocardial Lesion Formation during Laser Catheter Ablation in A Dog Model

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ABSTRACT. Monitoring of lesion formation during catheter ablation is difficult to accomplish. To achieve that goal, monitoring of intracardiac local electrograms (LEGs) was performed during laser application by using an open-irrigated electrode laser mapping and ablation (ELMA) catheter provided with pin electrodes at its tip in a dog model. A total of 112 laser impacts were aimed at the atrial (15 W/5–30 s, $n=60$) and at the ventricular free walls (20 W/20–50 s, $n=52$) in 12 dogs. Amplitudes of potentials in the LEG gradually dwindled and were abolished in 8–13 s (9.9 ± 1.6) and in 25–32 s (28.1 ± 1.9) after radiation. Lesions achieved were correlated with the LEG recorded during formation of each lesion. After permanent abolishment of potentials, lesions were transmural. Radiation limited to 3–5 s allowed for complete recovery of potential amplitudes and did not produce lesions. In contrast to that, continuation of radiation >5 s beyond 13 s or 35 s, occasionally caused myocardial and collateral damages or malignant arrhythmias. A light-guide protection system (LPS) and transesophageal light sensor (TLS) helped avoid such damage. During laser application LEGs recorded via the pin electrodes mounted at the ELMA catheter tip allowed for monitoring of lesion formation.

KEYWORDS. laser catheter ablation, monitoring of laser lesion formation, open-irrigated electrode laser mapping and ablation catheter, voltage of local intracardiac potential amplitudes.

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Introduction

Laser energy is light waves that are converted to heat to ablate and create scar tissue. It may be a safer and more effective heat-based energy than unipolar radiofrequency (RF) energy.^{1,2} Using laser energy, for lesion formation the catheter does not need pressure on the tissue,³ which

could mean it will have fewer complications than RF energy. Unlike other energy sources, contact force is not a factor in whether a laser lesion is transmural. In addition, laser energy can be adjusted when ablating tissues of varying thickness, with higher energy applied to thicker tissue and lower energy applied to thinner structures in the heart.⁴ A larger laser lesion enhances the prospects that lesions will be contiguous (without gaps) and could shorten procedure times. However, control of lesion formation in the myocardium during catheter ablation is still entirely in the experimental field.

In an effort to achieve this goal, intracardiac echocardiography⁵ and improvement of catheter stability by using robotic assistance were tested.⁶ Furthermore, to predict lesion size and to reduce or avoid the incidence of steam pop, a contact force sensor was incorporated in irrigated RF ablation catheters.⁷ However, not only lesion size but

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Figure 1: (a) Distal end of the electrode laser mapping and ablation catheter showing one of the two electrodes mounted on the outer surface of the tip. (b) Optical fiber tip creates a divergent light beam (vertical arrows). (c) The “donut”-like ring-shaped radiation field in front of the catheter endhole.

also steam pop and thrombus incidence depend strictly on the contact force exerted by the ablation catheter at the interface with cardiac tissue. An energy titration strategy with an endoscopic laser balloon ablation system was also tested.⁸ More recently, the feasibility of direct visualization of lesion formation was investigated by using magnetic resonance imaging-guided electrophysiology studies in humans.⁹

Methods

The study was performed in the Central Laser Laboratory and Experimental Animal Laboratory Facilities of the Helmholtz Center, Neuherberg, Germany, and in the Experimental Electrophysiology Laboratory, the University of Oklahoma, Health Science Center, Oklahoma City, OK, USA. The procedures followed were in accordance with institutional guidelines, and comply with the principles outlined in the Declaration of Helsinki. Animal experimental studies conformed to Directive 86/609/EEC on the protection of animals used for experimental and other scientific purposes, adopted in 1986 by the European commission, and complying with all applicable Laws and Regulations and Guidelines of the United States Food and Drug Administration regarding Good Laboratory Practice and Non-clinical Research (CFR Title 21, Parts 11 and 58). Institute Review Board approval was obtained.

Ablation procedure

Twelve healthy 12–19-kg beagles of either sex were anesthetized by intravenous thiamylal sodium 4%, 0.4 ml/kg and intubated for isofluran (0.8–1.5%) and nitrous-oxide anesthesia. Heart rate, quality of peripheral pulse, and atrial oxygen saturation were monitored throughout the procedure. After venous puncture in the right groin (Seldinger technique) the electrode laser mapping and ablation (ELMA) catheter RytmoLas (LasCor GmbH, Taufkirchen, Germany) was inserted via a steerable sheath (Agilis, MxT, St. Jude Medical, Inc. St. Paul, MN) and was manipulated in the heart under X-ray control. Continuous monitoring of bipolar intracardiac electrical potentials was

performed simultaneously with surface lead electrograms. Left heart access was achieved by using the transseptal laser puncture set TransLas (LasCor GmbH).¹⁰

The lumen of the ELMA catheter was filled via an infusion line prior to its insertion into the sheath with heparinized saline (5000 IU/l) and was flushed at a flow rate of 15 ml/min (continuous flow). During laser applications the flow rate was augmented automatically via the laser foot switch to 30 ml/min (“working flow”). For laser applications the catheter was brought in a stable position upon the target area but without pressure on the endocardial surface. Surface lead and Intracardiac LEGs were simultaneously and continuously monitored.

Equipment

The RytmoLas

The RytmoLas is an open-irrigated ELMA catheter, consisting of an 8F radiopaque (BaSO₄ 20%) plastic tube with a 400 μ m optical fiber fed into its central lumen and with two symmetrically arranged vertically positioned pin electrodes at its tip (Figure 1). The electrodes were connected to the manifold via cables running in the lumen of the catheter along with the optical fiber. During laser radiation the tip electrodes were riding symmetrically upon the illuminated field.

The transesophageal light sensor

The transesophageal light sensor (TLS) (SensoLas, LasCor GmbH, Taufkirchen, Germany) consists of a transesophageal probe, with a cylindrical light sensor 5 cm in length (“Ulbricht” tube) at its tip, which was positioned in the distal esophagus prior to the ablation procedures. The sensor was placed behind the posterior LA wall, where laser applications were aimed. The X-ray opaque tube of the SensoLas allowed for guiding of the laser impacts towards the esophagus. The outer end of the SensoLas was connected to an optometer (Gigahertz Optic GmbH, Tuerkenfeld, Germany) converting the detected laser light into power (Watts) that was measured by a power meter.

- 1 = Laser, 2 = Pump, 3 = Infusion line, 4 = Rotor, 5 = Safety box,
 6 = esophageal probe, 7 = Power meter, 8 = flow/bubble control
 9 = Luer connector, 10 = Laser catheter, 11 = Distributor and
 12 = electrode cables to the manifold for local electrograms (LEG)
- optical fiber for the laser light towards the heart, the same
 - - - optical fiber for the light-guide protections system (LPS)
 — infusion line for catheter irrigation flow through flowmeter
 - - - transesophageal light sensor (TLS) connected to the safety box
 -| -| - cable from the safety box to the laser

Figure 2: Schematic drawing of the esophageal monitoring system showing the laser (1), the peristaltic pump (2), safety box (5), the esophageal probe (6), and laser catheter (10) with their connections.

The safety box

This houses the optometer and the flowmeter. The flowmeter, an ultrasound flow control of the infusion line, ensured a continuous catheter irrigation flow of at least 5 ml/min. The box is connected to the laser and stops it automatically when the power registered by the transesophageal laser probe TLP exceeds the pre-set value or when the saline flow is below 5 ml/min or is inadvertently stopped, or the infusion liquid contains impurities or air bubbles.

The laser

The laser is a continuous wave (cw) 1064 nm Nd:YAG diode laser (MediLas 30 W 1064 D, LasCor GmbH, Taufkirchen, Germany) designed for cardiovascular laser applications. The laser was fitted with a LPS that can stop the laser automatically in case of optical fiber contact with tissue or blood. Contact will result in burning of tissue or blood that produces flashes of lightning at wavelengths other than the 1064 nm laser light. By retrograde sensing via the optical fiber, the

Figure 3: Local electrogram recorded via the pin electrodes of the ELMA catheter tip during laser application at 15 W/5 s aimed at the right atrial lateral wall (LEG RA), showing a gradual attenuation of atrial potential amplitudes with the start of radiation (A–A₁), and gradual recovery of potential amplitudes to their initial height after 20 s (A₁–A₂). I, II = surface lead electrocardiogram.

lightning stops radiation prior to the occurrence of thermal damage to the optical fiber tip or to the myocardium. The links of the safety chains are schematically displayed in **Figure 2**.

Study protocol

A total of 112 laser impacts were aimed at the atrial and the ventricular walls in 12 healthy dogs (8–12 per dog). Sixty impacts at 15 W/5–30 s were aimed at the right atrial (RA, n=34) posterior and lateral walls, and at the left atrial (LA, n=26) posterior wall adjacent to the esophagus. Four of the 34 RA impacts were limited to 5 s, and 30 impacts continued for 10, 20, and for 30 s (n=10, each). Two of the 26 LA impacts were also limited to 5 s, and 24 impacts continued for 10, 20, and 30 s (n=8, each). Lesions were placed in a vertical row at distances of 1.0–2.0 cm from each other. Fifty-two laser impacts at 20 W/20–50 s were aimed at various sites of the right (RV, n=28) and left ventricular (LV, n=24) free walls for 20, 30, 40, and 50 s (n=7 and n=6, each, respectively). Lesions were placed at distances of 2.0–3.0 cm from each other.

Hearts were removed after the experiments or after 3 months (n=2 dogs); lesions were 2,3,5-Triphenyltetrazolium chloride (TTC) or hematoxylin-eosin (HE) stained and examined morpho-histopathologically. Diameters of atrial lesions were measured on the epicardial side and maximum diameters of ventricular lesions were measured at a depth

where the widest distance was found. Sizes of lesions were correlated with the corresponding LEG registered during the formation of the same lesion.

Results

Laser effects on the atrial myocardium

Local potentials showed sharp spikes and monitoring of LEG was feasible also during laser application without noise. There was a gradual attenuation of local electrical potential amplitudes conspicuous in the LEG immediately with the start of radiation. After impacts limited to 5 s, voltages of amplitudes gradually reached their initial heights in 20–25 s (**Figure 3**), and lesions were not found in these areas. In contrast, after laser applications of 8–13 s (mean 9.9 ± 1.6), a permanent attenuation of atrial potential amplitudes was achieved (**Figure 4**). Consistent with these impacts, clear-cut circular transmural lesions were seen in both the right and the left atria without damage to the lungs or esophagus.

If radiation times of 20 s were attempted, the laser was stopped automatically after 15–17 s, when the preset power limit of the optometer was set to 1.5 W. In order to adhere to the study protocol for longer radiation times, the TLS was deactivated. RA lesions achieved after 20 s and 30 s of radiation were also transmural but produced collateral damage to the lung lobes. Similarly, laser impacts aimed at

Figure 4: (a) shows the surface lead ECGs I and II and a bipolar local atrial electrogram (LEG RA) recorded via the pin-electrodes of the ELMA catheter tip during laser application at 15 W aimed at the right atrial free wall. (b) shows the atrial lesion achieved during the procedure (circle). (c) shows the local left ventricular electrogram (LEG LV) recorded with the pin-electrodes of the ELMA catheter during laser application at 20 W aimed at the left ventricular free wall. (d) shows the lesion achieved during the procedure. Both the lesions were produced in dog hearts. LEGs show a gradual attenuation of the local potential amplitudes (A to A₁, and V to V₁) with the start of radiation and their permanent abolishment after 8 s and 22 s of radiation respectively (vertical arrows). Note: the clear-cut lesion of transmural coagulation necrosis without tissue vaporization and crater formation without intramural cavitation. The oblique arrow in (d) shows the assumed catheter orientation during laser application in the beating heart and points towards the translucent endocardial surface without thermal damage at the site of laser application.

the LA posterior wall adjacent to the esophagus produced transmural esophageal lesions (Figure 5).

Laser effects on the ventricular myocardium

Continuous attenuation of local potential amplitudes was always conspicuous in the LEGs with the start of laser

radiation. Occasionally, a few extrasystoles occurred during the first seconds of radiation (Figure 6). Subsequently, amplitudes dwindled continuously and eventually were abolished permanently after 25–32 s (mean 28.1 ± 1.9 s). Some of the lesions achieved after radiation times of 20 s and 30 s were near transmural (Table 1). After permanent abolishment of potential amplitudes on the endo- and epicardial surfaces, circular spots of coagulation

Figure 5: Collateral damage to the lung lobe, surface (a) and section view (b), and to the esophagus, external (c) and internal view (d), after laser application at 15 W/30 s aimed at the right atrial and left atrial posterior walls, respectively. Laser effects after various application times during LEG monitoring in the right ventricular free wall of dog hearts.

Figure 6: Electrocardiogram monitoring including intracardiac local electrogram (white middle tracing) during laser radiation aimed at the left ventricular free wall of a dog heart. (a) At the start (horizontal arrow) four polymorphic ventricular complexes are conspicuous. (b) The amplitudes of local potentials (V) dwindle gradually (vertical arrows), and (c) after 25 s were abolished permanently (vertical arrow).

necrosis were conspicuous. The lesions of homogeneous coagulation necrosis were clear cut: there was no tissue vaporization with crater formation (**Figure 4**). However, when radiation was continued for >5 s after permanent abolishment of local potential amplitudes in 9.6%, pop phenomenon with intramural cavitation occurred (**Figure 7**). Intramural cavitations or malignant arrhythmias were observed after 6–21 s (11.6 ± 5.5 s) of laser radiation continued after the abolishment of the potential amplitudes in the LEG (**Figure 8**).

Discussion

In our studies reported elsewhere we have shown that during laser application, continuous gradual attenuation

of amplitudes of local electrical potentials recorded via the ELMA RytmoLas catheter regularly occur.^{11,12} Based on these we have monitored local electrograms and have limited laser application times in our patients accordingly, from the very beginning of arrhythmia ablation.^{13–15} This study confirms a close correlation of the LEG and lesion formation. Primarily, monitoring of the local potential amplitude allowed for immediate verification of treatment efficacy. Subsequently, continuous dwindling of potentials reflects the spread of coagulation necrosis, the growth of the lesion in the myocardial wall. A permanent attenuation denotes the presence of irreversible effects, and abolition of local potential amplitudes suggests the achievement of a transmural lesion.

This relatively strong correlation between lesion formation and the local electrograms is mainly based on the

Table 1: Sizes of lesions (mm) achieved in dog hearts at various energy settings by using an open-irrigated electrode laser mapping and ablation (ELMA) catheter

Location of lesions and power (W)	Depth (transmural)	Diameter (maximum)			
RA (n=34) 15 W	1.5–5.7 (3.9 ± 1.5)	15 W/5 s (n=4) No lesion	/10 s (n=10) 3.2–7.0 (5.11 ± 1.3)	/20 s (n=10) 4.5–7.0 (5.63 ± 0.7)	/30 s (n=10) 4.2–9.0 (7.37 ± 1.5)
LA (n=26) 15 W	1.1–4.2 (3.1 ± 0.99)	15 W/5 s (n=2) No lesion	/10 s (n=8) 3.2–4.1 (3.68 ± 0.85)	/20 s (n=8) 3.9–6.1 (5.3 ± 0.7)	/30 s (n=8) 5.9–7.1 (6.6 ± 0.4)
RV (n=28) 20 W	3.8–8.4 (6.7 ± 1.45)	20 W/20 s (n=7) 5.0–8.1 (6.3 ± 1.06)	/30 s (n=7) 5.2–10.1 (7.9 ± 1.4)	/40 s (n=7) 6.6–10.5 (8.0 ± 1.35)	/50 s (n=7) 7.8–11.3 (9.3 ± 1.3)
LV (n=24) 20 W	Near transmural 8.0–14.1 (11.1 ± 1.9)	n=7/7 20 W/20 s (n=6) 4.6–9.1 (6.9 ± 1.2)	n=2/7 /30 s (n=6) 8.6–12.3 (8.45 ± 2.1)	/40 s (n=6) 9.7–12.9 (11.6 ± 1.1)	/50 s (n=6) 11.9–18.0 (15.6 ± 1.6)
			p=0.082	p=0.7047	p=0.2262
		Near transmural n=6/6	p=0.0050	p=0.075	p=0.0024

Figure 7: Laser effects after various application times during LEG monitoring in the right ventricular free wall of dog hearts.

geometry of the ELMA catheter tip with its two pin electrodes of 4/2 mm symmetrically overriding the illuminated field. Thus, electrical recordings are obtained from the endocardial area where lesion formation is

initiated and is gradually spreading concentrically. The increase in lesion is closely linked to the decrease in voltage of local electrical activity. Once the cardiac wall is completely devoid of electrical activity, transmural or

gray area: potential dwindle, white area: potentials are abolished

= episodes of ventricular fibrillation

= audible pops with intramural cavitations

Figure 8: Diagram illustrating the time intervals of the abolishment of potentials (gray area) and the time of the occurrences of collateral and myocardial damage.

near transmural coagulation can be assumed. The small and blunt local potentials occasionally still present after permanent attenuation of potentials represent rather far potentials.

With a permanent abolishment of local potentials the full laser effect is achieved. Continuation of radiation beyond this period of time is of no additional benefit. On the contrary, it may result in myocardial and collateral damage and compromise the good results of ablation. Timely stopping of radiation 3–5 s after abolishment of potentials can avoid these risks and has an important safety aspect.

Recovery of local potential amplitudes after radiation is stopped denotes a transient laser effect on the myocardium. The initially induced hyperemia, edema with swelling, and slight to moderate hemorrhagic infiltration of myocardium is reversible. Short laser impacts of 3–5 s are without a pathological correlate. Thus, monitoring of intracardiac LEGs during laser application can give important insights in the process of lesion formation and is helpful for the operator's routine work in the electrophysiology laboratory without the need of additional sophisticated equipment, although only an indirect control of lesion formation with the LEG laser ablation can be performed in a more controllable manner.

During laser ablation the catheter itself is not heated up. Photons penetrate deep intramurally and are frequently scattered before they are absorbed and heat is induced.^{16,17} Endocardial layers are translucent so that the laser light passes without absorption of photons and without generating heat. In addition, catheter irrigation at room temperature effectively cools the superficial layers of the endocardium.

By using the open-irrigated ELMA catheter, transmural lesions can be achieved at 15–20 W within seconds.¹⁸ Thus, the laser method may help cope with difficulties encountered in some patients with deep intramural ventricular or subepicardial arrhythmia foci or with arrhythmogenic substrates shielded in scarred myocardium not amenable to catheter ablation by using radiofrequency current or cryoablation, but susceptible to the deeper penetrating laser light.^{19–21}

More recently, the assessment of electrical activity and lesion efficacy was reported by using electrogram amplitudes recorded via pin electrodes near the ablation tip of a RF catheter.²² Endocardial voltage mapping technique was also used with a similar electrode arrangement to identified low-voltage right ventricular areas, which may represent the electroanatomical scar substrate of life-threatening tachyarrhythmias.²³ These reports support our experience with the LEG for monitoring of electrophysiological effects during catheter ablation and the feasibility of indirect visualization and monitoring of lesion formation in the myocardium during energy application.

Conclusions

Laser-induced attenuation of local electrical potential amplitudes in the LEG denotes the initiation of lesion formation and confirms the efficacy of energy application.

Subsequent constant gradual attenuation of amplitudes is consistent with the spread of coagulation necrosis, the growth of the lesion in the myocardial wall. After a radiation time limited to <5 s tissue effects are reversible, and amplitudes of potential will gradually regain their initial heights. Permanent abolishment of potential amplitudes suggests the achievement of a transmural lesion. Radiation continued after abolition of potentials will increase the risk of myocardial pop, malignant arrhythmias and the occurrence of collateral damage to the lungs and the esophagus. Monitoring of the LEG does not visualize directly the growth of lesions. However, as the ELMA catheter is magnetic resonance MR safe it would be intriguing to verify lesion visualization by producing laser lesions under MR control, preferably in a multicenter study trial for arrhythmia ablation in a larger number of patients. For the time being, LEGs represent for the operator in the electrophysiology laboratory a unique and simple method of monitoring myocardial lesion formation, and for the patient an unequaled safe and effective ablation procedure.

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